AFRL-RH-FS-TR-2022-0010

Review of Infrared Laser Induced Damage to the Crystalline Lens of the Eye

Nathaniel J. Pope
Brian J. Lund
SAIC

Chad A. Oian
711th Human Performance Wing
Airman Systems Directorate
Bioeffects Division
Optical Radiation Bioeffects Branch

10 August 2022

Interim Report for July 2022 to August 2022

Air Force Research Laboratory
711th Human Performance Wing
Airman Systems Directorate
Bioeffects Division
Optical Radiation Bioeffects Branch
JBSA Fort Sam Houston, Texas 78234

NOTICE AND SIGNATURE PAGE

This report was cleared for public release by the AFRL Public Affairs Office and is available to the general public, including foreign nationals. Copies may be obtained from the Defense Technical Information Center (DTIC) (http://www.dtic.mil).

"Review of Infrared Laser Induced Damage to the Crystalline Lens of the Eye "

(AFRL-RH-FS-TR- 2022- 0010) has been reviewed and is approved for publication in accordance with assigned distribution statement.

FERRIS.LYNDSEY.
MARIE.1381070391

Digitally signed by
FERRIS.LYNDSEY.MARIE.1381070391
Date: 2022.08.29 08:27:47 -05'00'

LYNDSEY M. FERRIS, Maj, USAF, BSC
Chief, Optical Radiation Bioeffects Branch

MILLER.STEPHANI
E.A.1230536283

Digitally signed by
MILLER.STEPHANIE.A.1230536283
Date: 2022.09.25 12:08:35 -05'00'

STEPHANIE A. MILLER, DR-IV, DAF
Chief, Bioeffects Division
Airman Systems Directorate
711th Human Performance Wing
Air Force Research Laboratory

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. REPORT DATE *(DD-MM-YYYY)*
10-AUG-2022

2. REPORT TYPE
Interim Technical Report

3. DATES COVERED *(From - To)*
July 2022 to August 2022

4. TITLE AND SUBTITLE
Review of Infrared Laser Induced Damage to the Crystalline Lens of the Eye

5a. CONTRACT NUMBER
FA8650-19-C-6024

5b. GRANT NUMBER

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)
Nathaniel J. Pope, Brian J. Lund, Chad A. Oian

5d. PROJECT NUMBER

5e. TASK NUMBER

5f. WORK UNIT NUMBER
H14B

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Air Force Research Laboratory
711th Human Performance Wing
Airman Systems Directorate
Bioeffects Division
Optical Radiation Branch
JBSA, Fort Sam Houston, Texas 78234

SAIC
4141 Petroleum Rd
Fort Sam Houston, Texas 78234

8. PERFORMING ORGANIZATION REPORT

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)
711th Human Performance Wing
Airman Systems Directorate
Bioeffects Division
Optical Radiation Branch
JBSA, Fort Sam Houston, Texas 78234

10. SPONSOR/MONITOR'S ACRONYM(S)
711 HPW/RHDO

11. SPONSOR/MONITOR'S REPORT NUMBER(S)
AFRL-RH-FS-TR-2022-0010

12. DISTRIBUTION / AVAILABILITY STATEMENT
Draft Distribution A. Approved for public release; distribution is unlimited. CLEARED: PA Case# AFRL-2022-4642. The views expressed are those of the author and do not necessarily reflect the official policy or position of the Department of the Air Force, the Department of Defense, or the U.S. government.

13. SUPPLEMENTARY NOTES

14. ABSTRACT

While there are many studies that examine the damage thresholds for the retina (and to a lesser extent the cornea), few studies examine laser damage to the lens, and even fewer of these include infrared (IR) wavelengths. The goal of this report is to summarize the published literature describing IR laser damage to the crystalline lens of the eye. A major conclusion of this report is that there is an unfortunate dearth of published information on IR laser damage to the lens. Only two studies include rigorous analysis of lens damage thresholds, and only at three select wavelengths (1318 nm, 1338 nm, and 1356 nm). One other rigorous threshold damage study is included as it reports no lenticular damage even with exposures powerful enough to ablate the cornea above. We have also collected scattered reports of lens damage due to IR laser exposure that do not determine thresholds (or often do not include key details of the exposure parameters), and studies of injury due to chronic exposure to broadband IR (glassblower's cataract).

15. SUBJECT TERMS

16. SECURITY CLASSIFICATION OF:
Unclassified

a. REPORT	**b. ABSTRACT**	**c. THIS PAGE**	**17. LIMITATION OF ABSTRACT**	**18. NUMBER OF PAGES**
U	U	U	U	11

19a. NAME OF RESPONSIBLE PERSON
Chad A. Oian

19b. TELEPHONE NUMBER *(include area code)*
(210) 539-8299

Standard Form 298 (Rev. 8-98)
Prescribed by ANSI Std. 239.18

This Page Intentionally Left Blank

TABLE OF CONTENTS

Section **Page**

List of Figures

1.0 SUMMARY

The development of infrared (IR) lasers has enabled a wide variety of military and civilian applications. Of particular interest for the military are the use of these lasers in targeting, range finding, communications, and even weapons systems. As the power of deployed IR lasers continues to rise, there has been an increasing need to assess the hazards posed by exposure. Injuries from IR laser exposure are most commonly found in the eyes and the skin. In general, the eye is more sensitive to laser damage for visible and near-infrared wavelengths than the skin, due to the refractive power of the eye focusing and concentrating light on the retina. While there are many studies that examine the damage thresholds for the retina (and to a lesser extent the cornea), few studies examine laser damage to the lens, and even fewer of these include IR wavelengths. The goal of this report is to summarize the published literature describing IR laser damage to the crystalline lens of the eye. A major conclusion of this report is that there is an unfortunate dearth of published information on IR laser damage to the lens. Only two studies include rigorous analysis of lens damage thresholds, and only at three select wavelengths (1318 nm, 1338 nm, and 1356 nm). One other rigorous threshold damage study is included as it reports no lenticular damage even with exposures powerful enough to ablate the cornea above. We have also collected scattered reports of lens damage due to IR laser exposure that do not determine thresholds (or often do not include key details of the exposure parameters), and studies of injury due to chronic exposure to broadband IR (glassblower's cataract).

2.0 INTRODUCTION

High-energy lasers have found increasingly frequent use in a wide variety of applications, including operational and battlefield contexts. Unlike a laboratory or other highly-regulated environment, where safety without any possibility of injury is the expected norm, a risk-based approach is more appropriate in these contexts. In this case, risk can be represented as the likelihood of exposure, relative to the severity of an injury due to that exposure. The overall outcome can thus be predicted and quantified using a dose-response model, relating the probability and level of exposure to the degree of potential damage. The research group here at the Bioeffects Division has extensive experience building and improving laser exposure dose-response models for over 15 years, incorporating hazard analyses relevant to both ocular and skin tissue [1-4].

Until recently, these models have focused on predicting threshold or onset-of-injury level of laser-tissue damage. These data have typically been gathered from *in vivo* studies using appropriate animal subjects, and thresholds are usually expressed in terms of the exposure needed to produce a minimum visible lesion (MVL). This is the most reasonable criterion standard when creating these models for use in well-regulated environments, where exposures can be limited to levels below the threshold, as then no injury will occur. However, as laser systems continue to increase in power, and these systems are deployed in outdoor environments, it has become more relevant to consider higher levels of possible damage (supra-threshold damage) for hazard and risk assessment.

When considering laser injury to the eye, three main anatomical structures can potentially be damaged. These are the cornea, the lens, and the retina. In the visible wavelength range the component with the lowest threshold for laser damage, and thus the most likely to be injured, is the retina. This is due to both the optical power of the eye focusing and concentrating light at the retina, as well as the intensely pigmented epithelium immediately underlying the photoreceptor cells, which strongly absorb light in the visible spectrum. In contrast, the cornea and lens are optically clear, and have extremely low absorption in this wavelength range. While ultraviolet (UV) wavelengths are absorbed and can cause damage to both the cornea and the lens [5], this report is focused on IR wavelengths, as this range has particular relevancy due to several recently developed and upcoming laser systems such as illuminator and designator lasers. IR wavelengths are absorbed highly by water. Both the cornea and lens contain high proportions of water, and absorb readily in the IR.

3.0 THE LENS

The crystalline lens of the eye is one of the most specialized structures in the body [6-8]. Its primary purpose is to allow variable refractive power, enabling the eye to focus on objects at varying distances [6, 8]. It is biconvex in shape, and flexible, allowing bending to occur, known as accommodation [7, 8]. These changes in curvature result in changes in the refractive power. The lens is composed of tightly organized, highly specialized cells. All of the cells in the lens are of the same cell type, following a developmental pattern as the lens develops and continues to grow with age [7, 8].

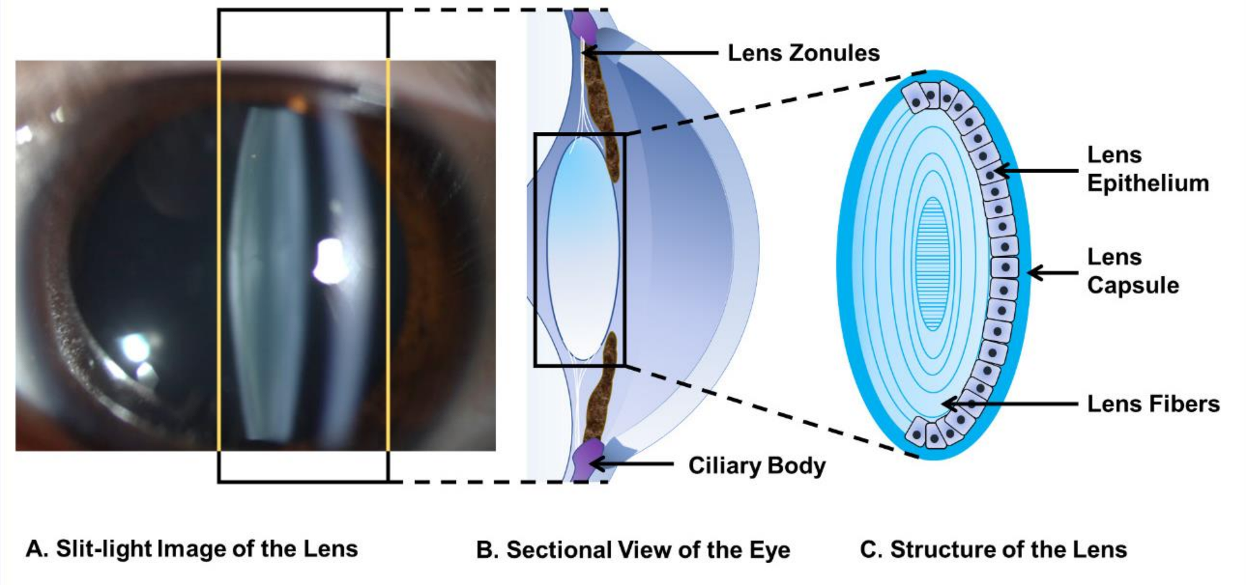

Figure 1. Diagram illustrating the overall structure of the lens and its location within the eye. From Ruan *et al*. [7], used with permission from BMJ Open Ophthalmology.

Cells begin as cuboidal epithelium in the germinative zone, a single layer of cells on the anterior surface [6]. These cells divide, and eventually migrate to the equator where they begin to elongate and invert to form lens fibers. During this process organelles such as mitochondria, golgi apparatus, rough and smooth endoplasmic reticulum, and eventually even the cell's nucleus, are degraded in order to better transmit light [6, 8]. During this process the cell membrane also is remodeled, increasing in density until it approaches that of the cytoplasm, which also decreases light scattering. Adjacent cells also form numerous interdigitations of their cell membranes, locking them together, and minimizing extracellular space between the cells, further aiding light transmission [7].

As the cells differentiate, the content of the cells also changes dramatically, and the cytoplasm is filled with an extremely high density of proteins, the crystallins. The protein content of lens cells is extremely high, representing as much as 60% of the total mass of the lens, and approximately 90% of these proteins are crystallins. The tight packing of these proteins also contributes to the optical properties of the lens, improving transparency and increasing the refractive index. If lens cell proteins are diluted to a lower concentration, light scattering increases [6, 8].

At birth the lens weighs approximately 65 mg, and rapidly increases in weight to approximately 160 mg by the age of 10. After this point, growth continues, but slows substantially, reaching up to about 250 mg by the age of 90 [6]. There is no turnover or replacement of cells as time goes on, so the cells present at birth remain in the center of the lens as an adult [7]. Because of this, the lens is particularly susceptible to damage over time. In addition to aging, UV light and oxidative stress can cause accumulating and permanent lenticular damage. Since crystallin proteins can also not turn over in nonnucleated fiber cells, these crystallins become increasingly chemically modified, and form aggregates or water insoluble structures [6, 8]. This, or other disruption of the fiber cells or their membranes, generally results in a loss of clarity, and increased scattering of light. This lens opacity, regardless of its cause, is commonly referred to as a cataract, and is the most frequent indication of lenticular damage. In the case of extreme supra-threshold exposures, the structure of the lens itself may be grossly disrupted with individual fibers separating, resulting in a severe loss of visual acuity [6, 8].

3.1 Infrared Laser Damage Studies And The Lens

The most significant conclusion of this report is that there is a minimal amount of systematic data regarding threshold or supra-threshold IR exposure of the lens. In fact, no studies were identified focusing specifically on IR lenticular damage thresholds, or supra-threshold dose-response. Rather, the few studies presented here are mostly examining the cornea and retina, and identified lens damage in the process.

Perhaps the most thorough study available is that by Zuclich *et al.* [9]. This study utilized a continuous wave Neodymium-doped Yttrium Aluminium Garnet (Nd:YAG) laser to produce either 1318 nm or 1356 nm to study ocular effects at supra-threshold energies on the cornea and retina. The corneal exposures also yielded threshold data for damage to the lens at these two wavelengths. Authors utilized an electronically controlled mechanical shutter to select exposure durations. Data was collected from two *in vivo* model systems: Dutch Belted rabbits and rhesus macaques. Prior to any laser exposure, the subjects were examined with a slit lamp, fundus camera, fluorescein angiography, baseline photograph taken at the fundus camera, and refraction to the nearest 0.25 diopter to establish a baseline. For corneal/lens exposures of rabbits, only the slit lamp and fundus camera were used during the examinations. For exposures to these anterior ocular tissues, the beam of the laser was focused to a 1.0 mm diameter for 1318 nm, and 0.7 mm diameter for 1356 nm [9-11].

At 1318 nm, subjects were exposed for times ranging between 0.2 s and 1 s when targeting the cornea and up to 20 s for the retina. Laser induced damage was observed in the cornea, lens, and iris of rhesus macaques, with retinal damage found as well in the rabbits. Damage thresholds for the lens were found to be substantially higher than those for the cornea. Indeed, in all cases that lenticular damage occurred, corneal damage was also found. Specifically, corneal thresholds were found to be 72 J/cm^2 in the rhesus macaques, and 175 J/cm^2 in the rabbits, versus 260 J/cm^2 for the lens (incident at the cornea) in both species [9].

Effects on the lens consisted of cataracts. In the case of threshold level exposures, the cataracts appeared as two discrete lesions; one directly below the cornea (anterior surface cataract) at the capsule or just sub-capsular, and another located directly behind but away from the capsule (cortical cataract), involving up to one third of the thickness of the lens. This posterior cataract appeared to show tapering of the lesion diameter with increasing depth. Unfortunately, the images included with the available digital versions of this article are poorly reproduced and lack nearly all detail. If the original images or underlying data can be located (as this study was conducted by the Optical Radiation Branch while at Brooks AFB) additional analysis may prove worthwhile. In the case of exposures slightly above threshold, the anterior cataract resolved within a few weeks while the cortical cataract persisted. Cataracts produced by twice or more than the threshold exposure remained for several months and were assumed to reflect permanent lens damage [9].

For 1356 nm exposures, the spot diameter (incident at the cornea) was reduced to 0.7 mm to increase the irradiance to values similar to those at 1318 nm. Again, the lenticular damage threshold was found to be substantially higher than that of the cornea. Specifically, the corneal threshold was 86.9 J/cm^2 in the rhesus macaques, and 58 J/cm^2 in the rabbits, while the lenticular damage threshold was ~ 2 x greater for rabbits and ~ 5 x greater for rhesus macaques. No retinal lesions were observed in either species for this wavelength. The appearance and temporal development of the lesions was similar to that of 1318 nm [9].

One other result of note was presented in this study. Specifically, laser irradiation of the iris could produce a cataract in the lens posterior to the irradiated region. The threshold for this cataract was substantially lower than that for the lens alone (approximately 50%). This was dubbed an “indirect cataract” as absorption appeared to occur in the iris, rather than directly by the lens. While this is somewhat outside the scope of this report, this effect is worth noting, as it may explain some of the results of the chronic broad spectrum IR induced injuries discussed in later sections [9].

The only other study identified which systematically investigated lenticular damage over a range of conditions was Jiao *et al.* [12] who measured the ED_{50} and extent of ocular damage induced by a single 5 ms pulse of 1338 nm laser light, over a range of spot size diameters. The *in vivo* model system used was New Zealand white rabbits, and the laser utilized was a pulsed Nd:YAG solid-state laser pumped by a Xenon lamp, producing a top-hat beam profile. Prior to exposures, corneas were examined using a slit lamp and retinas were examined using a fundus camera to establish the baseline. At 1 h, 6 h, 24 h, and 48 h post exposure, they examined these structures again. The results of this study were similar to those of Zuclich *et al.* [9]. In the corneal damage threshold level exposure, no lenticular damage was found. However, at approximately 1.5x the corneal ED_{50} value, 44.4 J/cm^2 (incident on the cornea) with a spot size of 1.91 mm, lenticular lesions at the anterior lens capsule appeared and were visible via slit lamp examination immediately following exposure. In contrast, exposures with intensity more than 5x the retinal ED_{50} did not result in any lenticular damage [12].

One additional study also bears mentioning here, specifically that of Berezin *et al.* [13]. Though it presents no actual lenticular damage, some useful conclusions can still be reached, as it examines a number of wavelengths further into the mid-IR range, and reports effects of irradiances substantially above the corneal damage threshold. Specifically, supra-threshold corneal exposures were reported at 1320 nm (Nd:YAG), 1540 nm (Glass:Yb-Er), 1960 nm ($BaYb_2F_8$:Er), 2090 nm (YAG-Cr-Tm:Ho), and 2840 nm (YLF:Er) utilizing chinchilla blue rabbits. For 1540 nm, 1960 nm, and 2840 nm exposures, the beam diameter was 2 mm, while for 1320 nm and 2090 nm the beam was passed into a 400 µm diameter silica optical to produce an approximately 400 µm diameter spot. Exposures at 1320 nm lasted for 150 µs while at all other wavelengths they lasted for 2 ms [13].

In the case of 1320 nm, no lenticular damage was detected. While this is somewhat surprising since lenticular damage was associated with supra-threshold corneal exposures at 1318 nm, and 1338 nm, the spot size and exposure duration in this study were substantially smaller and shorter than the previously mentioned studies. Additionally, the largest supra-threshold effect reported on these corneas was the formation of "grey cloudy opacity." At 4x the corneal threshold, damage to the iris was reported, but not to the lens [13].

In contrast, with 1540 nm experiments, energy levels were increased until "severe carbonization of epithelium, opacity of stroma and … intensive local scar and loss of corneal sphericity" occurred. Despite this extreme damage, no injury to the lens was observed. No lens damage was reported in the case of 1960 nm exposure either. However, this data is slightly less clear, as the supra-threshold effects on the cornea did not proceed past "intensive grey-white opacity" and "swelling and partially detachment of margin epithelium." With 2090 nm and 2840 nm exposures, supra-threshold effects were again dramatic, producing "tissue defect resembling a crater with appearance of carbonization" and "carbonization … and crater defect" respectively. In both cases, no lenticular damage was found [13].

Taken together, this data strongly indicates that exposures at wavelengths between 1540 nm and 2840 nm may not be capable of inducing lens damage (at least for the examined exposure times and spot sizes), due to the very high absorption of the cornea. The supra-threshold corneal injuries also result in opacification or carbonization, which further serves to reduce transmission to the lens. Alternatively, ablation of the cornea may also be dissipating substantial amounts of the energy, resulting in protection of the lens.

3.2 Other Reports of Infrared Laser Lens Damage

For the sake of completeness, we are also including an inventory of papers that do not appear to have a sufficient range of exposure parameters to assess, or which mention incidental laser-induced lenticular damage. Most of these papers are either case studies or experimental use of an IR laser for intraocular surgery, or are preliminary safety studies where either all exposure conditions produce damage, or it appears there is insufficient information to establish a clear trend. However, these studies may still prove useful as data-points, given the otherwise modest information available on this topic. These papers are presented in no particular order.

Krueger *et al*. present data that ultrashort pulse lasers at 1064 nm, 1057 nm, 1050 nm, and 780 nm with pulse widths from 15 ns to 150 fs and powers from 2.7 mJ to 1 µJ can all cause lenticular lesions. These appear to be laser induced breakdown lesions, rather than cataracts [14]. Pau *et al*. present data indicating that mode-locked Nd:YAG laser outputting 1-5 mJ in 1-5 bursts can induce lenticular disruption due to the formation of gas bubbles and separation of lens fibers. Specific laser exposure parameters are difficult to ascertain [15]. Vester *et al*. present data that mode-locked Nd:YAG at 1064 nm, with 3.2 mJ pulse energy, 30 ps pulses, in a 50 µm diameter spot, and Q-switched Nd:YAG at 1064 nm with pulse energies from 0.3 to 70 mJ, 12 ns pulse durations, and 60 µm diameter can induce lenticular lesions due to laser induced breakdown. These lesions are different in morphology but are clear ablations rather than cataractous [16]. Welch *et al*. reports that using a Q-switched Nd:Yag laser to perform iridiotomies on 13 eyes with one multimode burst of 6 pulses at between 6.5 and 7.5 mJ resulted in pitting of the lens in all cases [17]. Gwon *et al.* reports using a Q-switched Nd:YAG laser to deliver 6 to 97 laser spots of 2 to 8.3 mJ of energy and diameter of 50 µm, and a Nd:YLF laser at 1053 nm with 60 to 140 µJ per pulse to attempt to ablate normal and cataractous lenses. While the Nd:YAG laser produced opaque lesions, the Nd:YLF was reported to have ablated the lens, including the cataract, while leaving a clear lesion [18]. Gona *et al*. present data that Nd:Yag exposure at 3.9 to 4.2 mJ induced opaque lesions in the lens, with subsequent recruitment of epithelial cells which further opacified the region [19].

3.3 Wideband Infrared Lens Injury

That IR exposure can cause lenticular damage has been a well-known fact since long before IR lasers were invented. The first reported mention of the effects of IR on the crystalline lens was as early as 1739 [20, 21]. It was eventually connected to certain historical professions exposed to large amounts of heat in the 1800s, with cataracts associated with furnace works acquiring the name “glassblower’s cataract” since at least 1886 [20, 21]. However, whether this literature is relevant to modeling of IR laser lens damage is questionable.

An IR laser is generally a very narrow band source, and absorption occurs in the lens itself. In contrast, glassblower’s cataract is characterized by absorption of a wide range of wavelengths. Some studies have used black body sources [22, 23], others have focused on sources from 700 nm to 1400 nm [20], or 700 nm to 3000 nm [24], while others have used 3000 nm to 10000 nm [25]. Lastly, IR laser damage is generally an acute injury from a single exposure, or series of rapid pulses, while glassblower’s cataract is a chronic injury slowly developing over the course of years of low-level exposure.

Finally, though modernization of furnace design and ocular PPE has largely obviated the study of this pathology, a great deal of understanding has been gleaned. Though some early studies proposed that the lens could be a direct absorber of the harmful IR radiation, later studies seem to agree that this pathology is due to indirect absorption by the iris [20, 21, 23, 26, 27]. This is similar to the indirect cataract discussed in Zuclich *et al*. above [9]. As a result, it seems unlikely that this literature will be tremendously useful in the construction of IR laser lens dose-response models. However, some authors still put forward a direct lens absorption model [22, 24, 28], and references are provided for the sake of completeness.

4.0 CONCLUSIONS

In conclusion, while there several peer reviewed articles describing various IR induced lenticular injuries, many of them are of limited usefulness to dose-response modeling efforts. Only a handful of IR laser damage studies provide rigorous lenticular damage threshold data, and at a very limited number of wavelengths. Nevertheless, some preliminary conclusions can be made, if only due to inference when lenticular damage is not found. At wavelengths between 1300 nm and 1350 nm two studies reported it is possible to induce lenticular injury at energies that also induce supra-threshold lesions to the cornea [9, 12]. In contrast, Berezin *et al*. report that at all observed wavelengths between 1540 nm and 2840 nm, corneal absorption is so high that it is not possible to induce a lenticular injury [13]. Since supra-threshold exposures to the cornea in this range rapidly result in opacification (and eventually carbonization and ablation) [13], the lens is protected until nearly the point that the cornea becomes fully ablated.

Furthermore, multiple studies report that q-switched or mode-locked ultrashort pulse lasers in the NIR range around 1000 nm can also induce lenticular damage [14-19]. Unfortunately, none of these studies present damage threshold data, and many do not provide key exposure parameters, meaning their utility for modeling may be limited. Lastly, while IR exposure among glassblowers and furnace workers has been shown to cause lenticular damage [20-28], this is the result of chronic exposure to broad spectrum IR [20, 22-25], and is hypothesized to occur through an indirect mechanism [20, 21, 23, 26, 27]; thus the data from these studies may not be applicable to laser damage dose-response modeling efforts.

5.0 REFERENCES

1. E. M. Ahmed, E.A.E., D. F. Huantes, D. A. Wooddell, and R. J. Thomas, *A Probabilistic 1064nm Dose Response Model, Version 1.0*. 2009, Air Force Research Laboratory, Ft. Sam Houston, TX.
2. E. M. Ahmed, E.A.E., P. Kennedy, and R. J. Thomas, *"Human Laser Retinal Dose-Response Model,"*. 2018, Air Force Research Laboratory, Ft. Sam Houston, TX.
3. E. M. Ahmed, E.A.E., P. Kennedy, and R. J. Thomas, *"Human Variability in Laser Retinal Dose-Response Modeling,"*. 2018, Air Force Research Laboratory, Ft. Sam Houston TX.
4. E. M. Ahmed, E.A.E., B. J. Lund, and R. J. Thomas, *"Human Laser Skin Dose-Response Model,"*. 2020, Air Force Research Laboratory, Ft. Sam Houston TX.
5. Soderberg, P.G., et al., *Does infrared or ultraviolet light damage the lens?* Eye (Lond), 2016. **30**(2): p. 241-6.
6. Hejtmancik, J.F. and A. Shiels, *Overview of the Lens.* Prog Mol Biol Transl Sci, 2015. **134**: p. 119-27.
7. Ruan, X., et al., *The Structure of the Lens and Its Associations with the Visual Quality.* BMJ Open Ophthalmol, 2020. **5**(1): p. e000459.
8. Oyster, C.W., *The Human Eye: Structure and Function*. 2006: Sinauer.
9. Zuclich, J., et al. *Ocular effects of penetrating IR laser wavelengths*. in *Photonics West '95*. 1995. SPIE.
10. Zuclich, J., et al. *High-power lasers in the 1.3- to 1.4-um wavelength range: ocular effects and safety standard implications*. in *BiOS 2001 The International Symposium on Biomedical Optics*. 2001. SPIE.
11. Zuclich, J.A., D.J. Lund, and B.E. Stuck, *Wavelength dependence of ocular damage thresholds in the near-ir to far-ir transition region: proposed revisions to MPES.* Health Phys, 2007. **92**(1): p. 15-23.
12. Jiao, L., et al., *Ocular damage effects from 1338-nm pulsed laser radiation in a rabbit eye model.* Biomed Opt Express, 2017. **8**(5): p. 2745-2755.
13. Berezin, Y., et al. *Peculiarities of coagulation action of IR lasers (1-3 um) radiation on cornea*. in *Laser Optics '95*. 1996. SPIE.
14. Krueger, R.R., et al., *First safety study of femtosecond laser photodisruption in animal lenses: tissue morphology and cataractogenesis.* J Cataract Refract Surg, 2005. **31**(12): p. 2386-94.
15. Pau, H., et al., *Lesion and regeneration of the anterior and posterior lens capsule and cortex in rabbits Nd:YAG laser.* Graefes Arch Clin Exp Ophthalmol, 1989. **227**(4): p. 392-400.
16. Vester, C., et al., *Neodymium YAG laser effects on rabbit lenses. A scanning electron microscopic investigation using Q-switched and mode-locked lasers.* Graefes Arch Clin Exp Ophthalmol, 1984. **222**(2): p. 101-8.
17. Welch, D.B., et al., *Lens injury following iridotomy with a Q-switched neodymium-YAG laser.* Arch Ophthalmol, 1986. **104**(1): p. 123-5.
18. Gwon, A., et al., *Focal laser photophacoablation of normal and cataractous lenses in rabbits: preliminary report.* J Cataract Refract Surg, 1995. **21**(3): p. 282-6.
19. Gona, O., J.H. White, and L. Obenauer, *Wound healing by the rat lens after neodymium-YAG laser injury.* Exp Eye Res, 1985. **40**(2): p. 251-61.
20. Pitts, D.G. and A.P. Cullen, *Determination of infrared radiation levels for acute ocular cataractogenesis.* Albrecht Von Graefes Arch Klin Exp Ophthalmol, 1981. **217**(4): p. 285-97.
21. Lydahl, E., *Infrared radiation and cataract.* Acta Ophthalmol Suppl, 1984. **166**: p. 1-63.
22. Okuno, T., *Thermal effect of visible light and infra-red radiation (i.r.-A, i.r.-B and i.r.-C) on the eye: a study of infra-red cataract based on a model.* Ann Occup Hyg, 1994. **38**(4): p. 351-9.
23. Sisto, R., et al., *Infrared radiation exposure in traditional glass factories.* AIHAJ, 2000. **61**(1): p. 5-10.

24. Aly, E.M. and E.S. Mohamed, *Effect of infrared radiation on the lens.* Indian J Ophthalmol, 2011. **59**(2): p. 97-101.
25. Langley, R.K., C.B. Mortimer, and C. McCulloch, *The experimental production of cataracts by exposure to heat and light.* Arch Ophthalmol, 1960. **63**: p. 473-88.
26. Wolbarsht, M. *Damage To The Lens From Infrared*. in *1980 Technical Symposium East*. 1980. SPIE.
27. Wolbarsht, M., *The Origin of Cataracts in the Lens from Infrared Laser Radiation*. 1980, DUKE UNIV MEDICAL CENTER DURHAM NC.
28. Okuno, T., et al., *Cataract Formation by Near-infrared Radiation in Rabbits.* Photochem Photobiol, 2021. **97**(2): p. 372-376.